

Modern Concepts of Cardiovascular Disease

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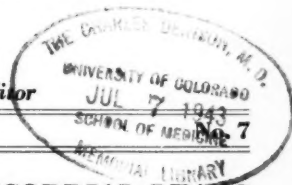
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THE VENTRICULAR COMPLEX IN MULTIPLE PRECORDIAL LEADS

PART II

Intraventricular block. Multiple precordial leads are of great value in the study of patients with intraventricular block and important advances in our knowledge of this disorder have resulted from a study of human precordial electrocardiograms and the corresponding tracings obtained by precordial and direct leads after cutting one of the branches of the His bundle in experimental animals.

In patients with left bundle branch block unipolar leads from the right side of the precordium yield QRS complexes dominated by a broad, often notched, downward wave. A small R-wave which reaches its peak very early in the QRS interval may or may not precede this downward deflection. The R-wave is inconspicuous or absent in these leads in left branch block because the interventricular septum is activated from right to left and the electrical forces produced by the free wall of the right ventricle are partly or completely neutralized. The large S wave is due to the relatively late activation of parts of the septum and the free wall of the left ventricle which makes the cavity of the right ventricle strongly negative throughout the QRS interval. In tracings from the left side of the precordium the QRS complex usually consists of a single bifid or slurred R wave but the transitional zone is often displaced to the left and R waves of the type mentioned may occur only in tracings from points at or beyond the left anterior axillary line. Since the cavity of the left ventricle is positive during the early phases of the QRS interval Q waves do not appear in tracings from the left side of the precordium in uncomplicated left bundle branch block. The part of the broad R wave which precedes the first distinct notch, often present on the ascending limb of this deflection, reflects the initial positivity of the cavity of the left ventricle while the portion of R which follows this notching, including the secondary elevation often seen, results from the late activation of the free wall of the left ventricle.

In precordial as in standard electrocardiograms showing bundle branch block, the T waves are usually opposite in direction to the chief deflection of the QRS complex consequently when left branch block is present, the T wave is ordinarily upright in tracings from the right side of the precordium and inverted in those from the left side.

In patients with right bundle branch block the QRS complex of the leads from the right side of the precordium is characterized by the presence of an initial wave, R, of variable size, followed by a downward deflection which may or may not extend below the baseline. This deflection is followed in turn by a very late and usually prominent broad upstroke, R'. The latter is due to the late and unopposed activation of the free wall of the right ventricle beneath the electrode while the preliminary upward deflection R represents forces produced by activation of

the interventricular septum from left to right which give rise to initial positivity of the cavity of the right ventricle. Thus in right branch block QRS complexes of an M shape with the second upstroke of the M higher than the first are characteristic of records obtained from the region of the precordium overlying the right ventricle. As the exploring electrode is shifted farther to the left, the preliminary wave, R, increases in size while the second upstroke, R', decreases. Leads from the left side of the precordium yield complexes with a prominent, narrow, upward R deflection followed by a broad S wave. In these leads a small Q wave is often present.

Precordial electrocardiograms are often helpful when the QRS interval in standard limb leads lies between 0.10 and 0.12 second and for other reasons the presence of incomplete bundle branch block is suspected. In incomplete left branch block the tracings obtained with multiple unipolar precordial leads are identical with those obtained in complete left branch block as regards the earliest components of the QRS complex but may be difficult to distinguish from those that represent left ventricular hypertrophy. The presence of a Q deflection in the leads from the left side of the precordium makes the presence of block extremely unlikely, but the absence of such a deflection has no important differential significance. In incomplete right branch block the earliest QRS deflections do not differ from those that characterize complete right branch block, but the QRS interval is shorter and the secondary, R', waves of the leads from the right side of the precordium are much smaller.

Myocardial infarction. Precordial electrocardiograms often display characteristic signs of myocardial infarction when limb leads do not, and furnish important data bearing upon the location and size of the region of muscle involved.

Much of our knowledge concerning the electrocardiographic changes produced by myocardial infarction has been obtained by the use of unipolar direct leads in experiments upon animals. Immediately after ligation of the artery which supplies the region upon which the exploring electrode rests the RS-T junction is displaced upward. This effect is ascribed to acute injury to the ventricular muscle deprived of its blood supply. It is accompanied or followed by a pronounced decrease in size or complete disappearance of the R wave normally present. The third modification of the ventricular complex consists of sharp inversion of the terminal portion of the T deflection which first appears when the elevation of the RS-T junction begins to subside and is often well developed before this junction has reached its normal position. It is of much greater duration than the RS-T displacement but usually disappears eventually. In experimental animals these secondary T wave changes are much more conspicuous in leads

from the peripheral than in leads from the central portions of the infarct and they are apparently due to prolongation of systole in a zone of subepicardial muscle surrounding the region within which the myocardium is dead. This explains why typical "coronary" T waves are often recorded from a precordial region that is larger than or not coextensive with the area which yields characteristic changes in the QRS complexes.

The peculiarities of the QRS complex in direct leads from an infarcted region are due to the transmission of the potential variations of the ventricular cavity to the epicardial surface of the region involved. The potential of this surface can differ materially from that of the adjacent cavity only when the intervening muscle is producing an electromotive force. When intraventricular conduction is normal both ventricular cavities are electrically negative throughout the QRS interval, and large QS deflections therefore appear in direct leads from transmural infarcts. When the infarcted region contains a considerable amount of muscle capable of responding to the cardiac impulse the bulk of this muscle is usually in the outer layers of the ventricular wall. Under these circumstances the potential of the ventricular cavity is well transmitted to the epicardium during the earlier and sometimes during the last parts of the QRS interval and the QRS complex consists of an abnormally large Q wave and a subnormal R deflection or has an outline similar to the letter W.

The potential variations of the epicardial surface of the infarcted region are transmitted to the adjacent parts of the body. If the infarct involves the ventral wall of the heart they are directed toward the precordium and characteristic changes in the QRS and T complex appear in precordial leads. If the lateral surface of the left ventricle is also involved they are transmitted to the left arm and similar changes appear in lead I. If the diaphragmatic wall of the heart is infarcted they are transmitted to the lower third of the esophagus, the back and the left leg. Distinctive signs then appear in esophageal leads and in leads II and III.

The electrocardiogram can be regarded as diagnostic of infarction only when typical changes in the QRS complex are accompanied by RS-T displacement or "coronary" T waves. On the basis of the leads in which these changes appear a variety of infarcts may be recognized. In some cases of anterior infarction characteristic modifications in the QRS complex are present for a very short period or do not develop, and only RS-T displacement followed by sharp inversion of the T waves is observed. Anterior infarcts which produce no lasting modifications of the QRS complex in either precordial or limb leads are small or incomplete and seldom lead to serious complications. On the other hand infarcts which give rise to diagnostic signs in all or in all but one of the six precordial leads are extensive and serious. Infarction of the antero-septal wall of the left ven-

tricle produces typical changes in the ventricular complex of the leads from the right side of the precordium but none in lead I. The latter lead seldom shows more pronounced signs of infarction than lead V, which is not affected by antero-septal infarction or displays abnormal T waves only. Antero-lateral infarction gives rise to characteristic changes in leads from the left side of the precordium and in lead I.

When the posterior or diaphragmatic wall of the left ventricle is involved typical changes appear in leads from the lower levels of the esophagus but precordial leads seldom yield data that have important diagnostic significance. In some instances extremely tall T waves occur in these leads and the R waves recorded from points near the sternum may be unusually large. Postero-lateral infarcts often produce abnormally large Q waves in the leads from the extreme left side of the precordium and give rise to inverted T waves in lead I and prominent Q waves in leads II and III. Antero-septal infarcts may produce similar changes in the standard leads. Postero-inferior infarcts occasionally produce diagnostic changes in a lead from the tip of the ensiform cartilage without having the same effect upon the ventricular complexes of the other precordial leads.

The presence of left bundle branch block prevents the occurrence of diagnostic signs of infarction in both the precordial and the limb leads. It induces initial positivity of the cavity of the left ventricle which makes impossible the appearance of QS deflections or abnormally large Q waves. The T waves are so greatly modified by the conduction defect that "coronary" T waves rarely occur. It is seldom possible, therefore, to make an electrocardiographic diagnosis of infarction under these circumstances. When right bundle branch block is complicated by infarction the situation is quite different. The cavity of the left ventricle is then negative throughout the QRS interval, as when intraventricular conduction is normal. Typical signs of infarction appear in the precordial leads but usually not in the limb leads when the ventral wall of this chamber is involved, and in leads II and III when its posterior wall is affected. These changes in the QRS complex are also accompanied by characteristic modifications of the T wave.

When antero-septal infarction is followed by postero-lateral infarction, there is a tendency for the QRS changes produced by the former to disappear or become less typical. Under these circumstances R waves may reappear in leads from the right side of the precordium and typical or semi-characteristic modifications of the ventricular complexes of the fifth and sixth precordial leads appear for the first time.

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